VIA MEDICA

PRACA KAZUISTYCZNA

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Lung abscess due to *Streptococcus pneumoniae*: a case series and brief review of the literature

Ropień płuca wywołany przez *S. pneumoniae*: opis przypadków i przegląd literatury

The authors declare no conflict of interest

Abstract

Anaerobes used to be the most common cause of community-acquired lung abscess, and *Streptococcus* species used to be the second most common cause. In recent years, this has been changing. *Klebsiella pneumoniae* is now an increasing cause of community-acquired lung abscess, but *Streptococcus* species continue to be major pathogens. Necrotizing pneumonia has generally been regarded as a rare complication of pneumococcal infection in adults. Type 3 Streptococcus pneumoniae was the single most common type implicated in necrosis; however, many other serotypes were implicated. This entity predominately infects children, but is present also in adults. Lung abscess in adults due to *Streptococcus pneumoniae* is not common. In this regard we present a case series of pulmonary cavitation due to *Streptococcus pneumoniae* and discuss the possible pathogenic mechanism of the disease.

Key words: lung abscess, *Streptococcus pneumoniae*, pathogenic mechanism

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Streszczenie

Bakterie beztlenowe stanowiły w przeszłości najczęstszy czynnik etiologiczny pozaszpitalnego ropnia płuca, *Streptococcus* był na kolejnej pozycji pod względem częstości. W ostatnich latach obraz ten uległ zmianie. Wzrosła liczba przypadków pozaszpitalnego ropnia płuca spowodowanego przez *Klebsiella pneumoniae*, nadal kolejnym co do częstości jest *Streptococcus species*. Martwicze zapalenie płuc jest rzadkim powikłaniem infekcji pneumokokowej u dorosłych. W tych przypadkach najczęściej izolowano serotyp 3 *S. pneumoniae*, jednak inne serotypy mogą również być przyczyną martwicy. Zakażenie serotypem 3 występuje głównie u dzieci, może być jednak zdiagnozowane również u dorosłych.

Ropień płuca u dorosłych wywołany przez szczep *S. pneumoniae* nie jest powikłaniem częstym. Z tego powodu w obecnej pracy przedstawiono serię przypadków, w których stwierdzono naciek płuca z rozpadem i wyhodowano *S. pneumoniae*. Omówiono także możliwe patomechanizmy choroby.

Słowa kluczowe: ropień płuca, *Streptococcus pneumoniae*, patomechanizm choroby

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Introduction

Anaerobes used to be the most common cause of community-acquired lung abscess, and *Streptococcus* species used to be the second most common cause of this problem. In recent years, this has changed. *Klebsiella pneumoniae* is now an increasing cause of community-acquired lung abscess [1], but *Streptococcus* species remain major pathogens. In a recent retrospective review of 205 patients, Takanayagi et al. [1] documented 122 bacteriological results, with 189 bacterial species isolated. Pure aerobic, mixed aerobic and anaerobic, and pure anaerobic bacteria were isolated in 90 (73.8%), 17 (13.9%) and 15 (12.3%) patients, respectively. The four most common aetiological pathogens were *Streptococcus* species 141 (59.8%), anaerobes (26.2%), *Gemella* species (9.8%) and *Klebsiella pneumoniae* (8.2%). *Streptococcus mitis* was the most common among the *Streptococcus* species. *Streptococcus pneumoniae* was found in three patients (2.12%) [1]. Necrotizing pneumonia has generally been regarded as a rare complication of pneumococcal infection in adults [2, 3]. Serotype 3 of *Streptococcus pneumoniae* was the single most common type implicated in necrosis; however, many other serotypes were implicated [2]. This entity predominately infects children, but is present also in adults (Table 1) [2]. In the USA and in Taiwan, infection with *Streptococcus pneumoniae* serotype 3 (mainly ST180) was a significant contributor to necrotizing pneumonia and lung abscess [3]. In Taiwan, serotype 14 was another common serotype that caused necrotizing pneumonia. However, there is no monopoly among the serotypes; other serotypes, including 6B, 19F, 23F and 19A, at a lower frequency, may also cause necrotizing pneumonia [4]. In a retrospective review conducted by Yen et al. covering twenty years (1982–2002), 23 patients presented with a lung abscess; the most common micro-organism isolated was again *Streptococcus pneumoniae* [5]. Lung abscess in adults due to *Streptococcus pneumoniae* is not common, but it is not rare. In this regard we present a case series of pulmonary cavitation due to *Streptococcus pneumoniae* observed in our departments in the last two years.

Case series

Case 1

A 45-year-old man with a medical history that included only intermittent bronchial asthma was admitted to the hospital with a 2-month history of productive cough, purulent sputum, low-grade fever, intermittent chest pain, asthenia and anorexia with accompanying weight loss (5 kg). He also complained of haemoptysis with an onset two days before presentation. On examination, the patient appeared ill, but not toxic: his temperature was 37°2, heart rate 78 bpm, respiratory rate 18 breaths/min, arterial blood pressure 105/70 mm Hg and SaO_2 was 99% on room air.

Bronchial breath sounds were noted over the area of the right upper lung along with scattered crackles. Laboratory evaluation demonstrated a WBC count of 8540 cells/μl, with 74.5% neutrophils and 13.0% lymphocytes; RBC was 4,770,000 cells/μl; haemoglobin 14.0 g/dL; PLT count 283,000 cells/μl; C-reactive protein 3.51 mg/dL; LDH 255 U/l and total protein was 7.44 g/dL with 17.0% γ-globulins. A chest radiograph showed consolidation with a cavitation inside at the right upper lobe (Fig. 1A). Chest computed tomography (CT) showed a spherical cavitary pulmonary mass of 6.7 × 4.6 cm with air-fluid level at the level of the dorsal segment of the right upper lobe (Fig. 1B). Flexible bronchoscopy revealed bronchial oedema with narrowing of the right lobar superior bronchus. Moreover, a small amount of purulent secretion was seen. A cytological examination showed inflammatory cells but no malignancy. Direct bacterioscopy and bacteriological analysis (cultures and polymerase chain reaction — PCR) performed on bronchoalveolar lavage and bronchoaspirate were negative for *Mycobacterium* tuberculosis. Because of the suspicion of an underlying malignancy, a CT-guided biopsy was performed in the right upper lobe.

The cytological examination showed inflammatory cells compatible with active inflammation, but no malignant cells were seen. A Gram-stained smear of sputum revealed numerous polymorphonuclear neutrophils and gram-positive cocci in pairs and chains; the culture eventually yielded *Streptococcus pneumoniae*. All the blood cultures collected upon admission were negative, as was the *Streptococcus pneumoniae* urinary antigen. A dental radiograph and a CT of nasal and paranasal sinuses did not show infectious foci. The minimum inhibitory concentrations breakpoint of penicillin G, ceftriaxone, erythromycin and imipenem were ≤ 0.06, ≤ 0.06, ≥ 0.5 and ≤ 0.03, respectively. The biochemical and susceptibility profile of these strains was assayed using an automated system for identification and susceptibility tests (ViteK Bio Merieux SA, France). Criteria to define susceptibility or non-susceptibility were based on the Eucast guidelines (www.eucast.org/clinical_breakpoints). The patient was treated with Amoxicillin/Clavulanate 2000 mg/200 mg IV every 8 hours for three weeks.The patient contin-

Table 1. Cases of necrotizing *pneumococcal pneumonia* and lung abscess

Reference	Subjects	N° of cases	Blood or pleural culture	N° of cases type/ /Pneumococcal serotype	Outcome
Danner 1968	Adults	2	Not done	1/serotype 7	1 death
Proctor 1977	Adults	1	Positive	not typed	alive
O'Reilly1978	Adults	1	Positive	not typed	alive
Yangco 1980	Adults	4	Positive	1/serotype 3 3/not typed	alive
Leathermann 1984	Adults	1	Positive	not typed	alive
Isaacs 1986	Adults	2	Positive	1/serotype 8 1/serotype 23	alive
Hammond 1993	Adults	1	Positive	1 not typed	alive
Kerem 1994	Children	4	Positive	4 not typed	alive
Donnelly 1998	Children	8	Not specified	8 not typed	alive
Hoffer 1999	Children	6	One Positive	6 not typed	alive
McCarthy 1999	Children	3	Positive	3 not typed	alive
Wong 2000	Children	3	One Positive	3 not typed	alive
Hodina 2002	Children	3	Not done	3 not typed	alive
Yu 2003	Adults	4	Positive	4 not typed	not specified
Kosucu 2004	Children	7	Positive	7 not typed	alive
Hsieh 2004	Children	13	Positive	13 not specified	not specified
Hsieh 2006	Children	14	Positive	5/serotype 14 2/serotype 3 1/serotype 6A 1/serotype 18C 5 not typed	alive
Ramphul 2006	Children	13	Positive	4/serotype 1 3/serotype 3 2 serotype 14 2/serotype 9 2 non typed	alive
Bender 2008	Children	33	Positive	11/serotype 3 4/serotype 19 4 /serotype 19A 3/serotype 1 2/serotype 6b 1/serotype 4 1/serotype 6A 1/serotype 7 1/serotype 19F 2/not typable 3 not typed	1 death
Fretzayas 2008	Children	10	Positive	10 not typed	alive
Sawicki 2008	Children	18	Positive	18 not typed	alive
Kalaskar 2009	Children	4	Positive	4/serotype 19A	alive

ued the amoxicillin/clavulanate (875 mg/125 mg tablets every 8 hours) after the discharge for two weeks. After six weeks a chest CT showed a complete resolution of the lung abscess.

Case 2

A 27-year-old man with a medical history that included no previous diseases was admitted to the hospital with a 1-month history of productive cough, light fever, asthenia, anorexia and severe weight loss (30 kg). At admission, his temperature was normal (36.5°C); on examination the patient appeared ill but not toxic, his temperature was 37°2, heart rate 98 bpm, respiratory rate 18 breaths/ /min, blood pressure 100/66 mm Hg, and SaO_2 was 97% on room air. Bronchial breath sounds were noted over the right upper lung along with scattered crackles. Laboratory evaluation demonstrated a WBC count of 10,700 cells/μl, with 75.5% neutrophils, 14.1% lymphocytes; RBC 4,140,000

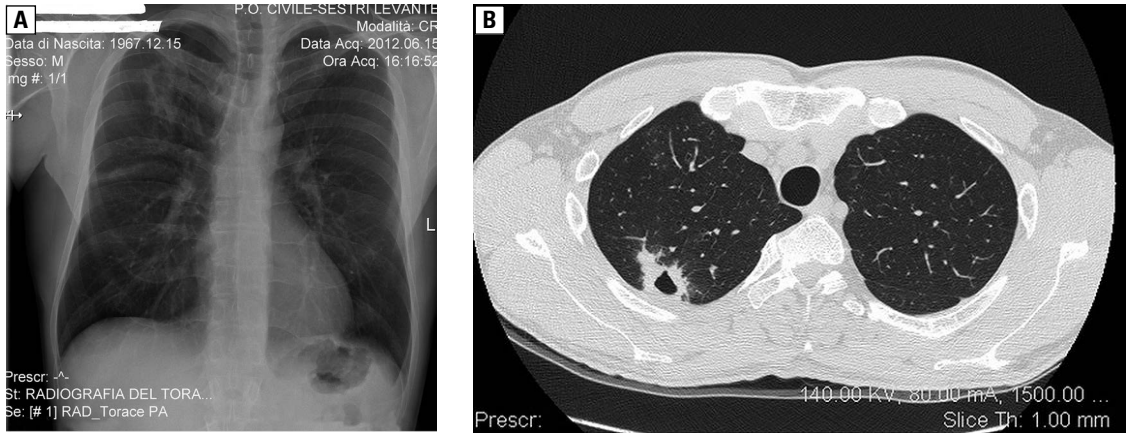

Figure 1. A — chest radiograph shows a right upper fluid-filled cavitation; **B** — chest computed tomography: spherical mass with air-fluid level (6.7 × 4.6 cm) at the level of the dorsal segment of the right upper lobe

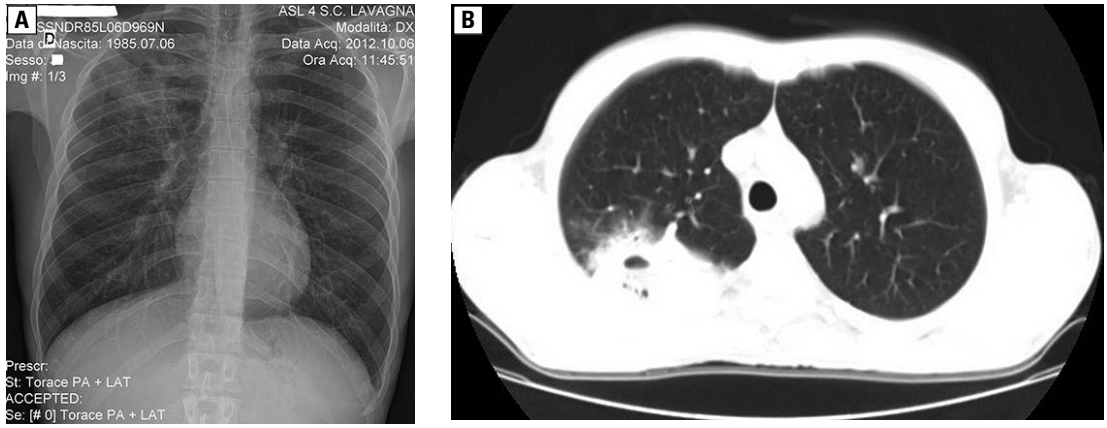

Figure 2. A — chest radiograph shows a right upper cavitation; **B** — chest computed tomography: cavitary pulmonary lesion (10.7 × 7.9 cm) at the level of the dorsal segment of the right upper pulmonary lobe

cells/μl, haemoglobin level of 12.4 g/dL; PLT count of 241,000 cells/μl, C-reactive protein 8.05 mg/ /dL, LDH 201 UL, total protein 7.31 g/dL with 15.1% of γ-globulins. A chest radiograph showed an inhomogeneous opacity with blurred edges and irregular extension involving the right upper lobe with small cavitations inside (Fig. 2A). Chest CT showed a large pulmonary consolidation (10.7 × 7.9 cm) with areas of cavitation at the level of the dorsal segment of the right upper lobe (Fig. 2B). The patient underwent bronchial fibrobronchoscopy: bronchial oedema with narrowing of the bronchial lobar superior and a small amount of purulent secretion was seen. A cytological examination showed inflammatory cells but no malignancy. All tests performed as in the previous case were negative for *Mycobacterium tuberculosis*.

A Gram-stained smear of bronchial aspirate revealed numerous polymorphonuclear neutrophils and gram-positive cocci in pairs and chains; the culture eventually yielded *Streptococcus pneumoniae*. The MIC were identical to those of the previously described case. All the blood cultures collected upon admission were negative, as was Streptococcus pneumoniae urinary antigen. A dental radiograph and a CT of nasal and paranasal sinuses did not show infectious foci.

The patient was treated with Amoxicillin/ /Clavulanate 2000 mg/200 mg every 8 hours for three weeks and after the discharge with Amoxicillin/Clavulanate (875 mg/125 mg tablets every 8 hours) for a further three weeks. After eight weeks a CT of the chest showed complete resolution of the lung abscess.

Case 3

A 35-year-old homeless man, presented in February 2012 at the emergency unit, with a 2-day history of fever, chest pain and dyspnea. He had lived in Spain for 3 years. The patient was a smoker with a history of alcohol abuse. On admission his respiratory rate was 40 breaths/min,

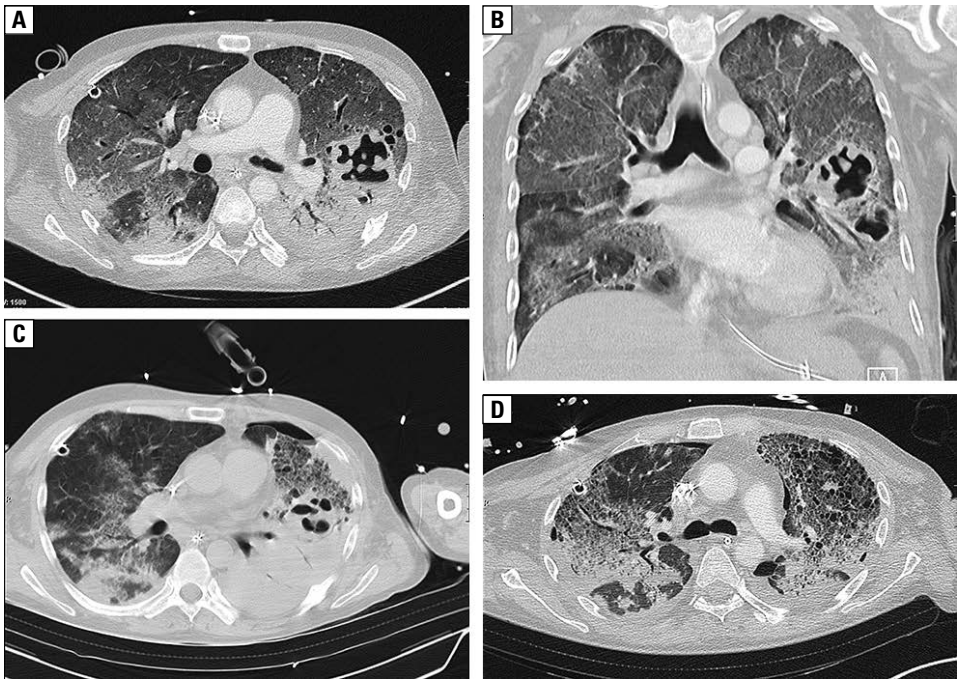

Figure 3. A — chest computed tomography (coronal reconstruction) diffuse bilateral ground-glass opacities; multiple small diffuse patchy consolidations in the lungs. A large dense opacity at the lower left pulmonary lobe. Two cavitations (abscesses) with thick walls and irregular inner edges in the medium-basal zone of the left pulmonary lobe; **B** — chest computed tomography (axial reconstruction) pulmonary dense opacities associated with reticular opacities, small pleural effusion and small pneumothorax in the left pulmonary lobe. Diffuse patchy opacities associated with ground-glass opacities and small pleural effusion; **C** — chest computed tomography: small pneumothorax, reduction of pulmonary consolidation. Ground-glass opacities associated with bullous-cystic areas in the left lower lobe. Ground-glass opacities associated with patchy opacities and small pneumothorax in the right lower lobe

SaO_2 92%, temperature 38.8^{0}C, blood pressure 155/110 and heart rate 140 bpm. On examination he was cyanotic with lividity. Lung auscultation revealed left-sided crackles. The laboratory studies on admission to ICU revealed elevated C-reactive protein 22.1 mg/dL, WBC 14000 cells/μl with neutrophils 84% and lymphocytes 6%, indicating bacterial infection. Chest X-ray and CT showed diffuse bilateral pulmonary ground-glass opacities, multiple small patchy consolidations in both lungs and a large consolidation with two cavitations in the left lower pulmonary lobe (Figs 3A, B). Blood cultures, urinary antigen (*pneumococcus and legionella pn.*) and nasopharyngeal swab for respiratory viruses were taken and empirical therapy with levofloxacin 500 mg/12 h plus ceftriaxone 1 g/24 h were established. In addition, therapy with oseltamivir and trimetoprim-sulfamethoxazole was added; trimetoprim-sulphamethoxazole was stopped when the results of HIV were negative. The patient was isolated for prevention for influenza virus; on the 2nd day the test for influenza virus was noted to be negative and the patient was removed from the isolation room. Because of worsening of the clinical and respiratory conditions, the patient required initial ventilator settings with FiO_2 100%, positive end-expiratory pressure (PEEP) 16 cm H_2O and tidal volume 460 mL. The initial antibiotic treatment was modified for levofloxacin plus meropenem. The diagnosis of the patient was septic shock, bilateral pneumonia with severe respiratory failure. The aetiology of this severe illness seemed to be Streptococcus pneumoniae. Blood culture collected in the emergency department on admission and urinary antigen were positive for *Streptococcus pneumoniae*, the strain was multi-sensitive and the serotype identified was 9V, nasofaringeal swab for respiratory viruses and antigen test for *L. pneumophila* were negative. Culture of tracheo-bronchial aspirate (BAS) on day 2 was positive for pneumococcus. On the 5th day, blood cultures were positive for *S. pneumoniae* plus *Streptococcus viridans*. Therefore, antibiotic therapy was adjusted to meropenem plus vancomycin plus levofloxacin. Chest CT scan on the 9th day revealed dense opacities associated with reticular opacities in the left lobe; moreover, small bilateral pleural effusion, a small left pneumothorax and bilateral, patchy consolidations associated with ground-glass opacities (Fig. 3C) were noted.

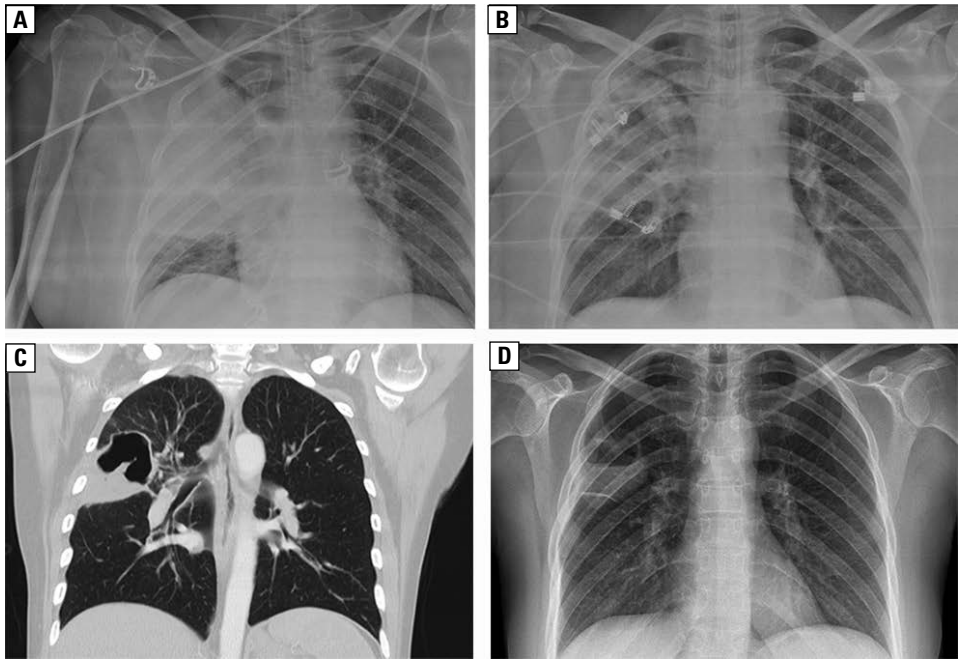

Figure 4. A — chest X-ray. Large consolidation in medium-upper zone of the right lung; **B** — chest X-ray. Large consolidation and cavitation inside; **C** — chest computed tomography (coronal reconstruction) at day 18: cavitation with thick walls and irregular inner edges; **D** — chest X-ray at discharge: further reduction of the cavitation in the upper pulmonary lobe

On the 14th day, another chest CT scan was performed, revealing enlargement of the bilateral pleural effusions and bilateral small pneumothoraces, as well as a reduction of pulmonary consolidation and ground-glass opacities associated with bullous cystic areas in the left lower lobe (Fig. 3D). Owing the severity of the respiratory failure, on the 16th day tracheotomy was performed and invasive mechanical ventilation continued. For persistence of abundant secretions, a tracheo-bronchial aspirate was performed on the 24th day with results positive for *Pseudomonas aeruginosa* resistant to ciprofloxacin and imipenem. Chest radiography showed impressive atelectasis. The antibiotic regimen was again altered to ceftazidime plus amikacin. On the 32nd day the patient achieved clinical stability and invasive ventilation was stopped.

Chest X-ray showed bilateral interstitial opacities and a dense opacity in the middle-lower field of the left lung. New culture of BAS was negative. On the 36th day the patients was transferred to the ward and was discharged on the 42nd day.

Case 4

We describe a 49-year-old woman, who presented in January 2010 at the emergency department with a 4-day history of fever, vomiting and diarrhoea; the night before coming to the hospital she experienced syncope at home. The emergency services intubated the patient before arriving at hospital because of life-threatening hypoxia (SaO_2 < 50% on FIO_2 100%). She did not have toxic habits, and the family said that she had borderline personality disorder and suffered from recurrent otitis. On admission she had Glasgow Coma Scale 10 and nuchal rigidity was present. The laboratory studies on admission revealed elevated C-reactive protein 27.2 mg/dL, WBC 19000 cells/μl with neutrophils 76% and lymphocytes 3.3%.

Chest X-ray showed a subtotal consolidation of the right lung with sparing of the basal and apical segments (Fig. 4A). Cerebrospinal fluid, blood cultures and tracheo-bronchial aspirate were obtained. Empirical therapy with meropenem plus vancomycin was established. She was admitted to the ICU immediately. Cranial CT revealed possible inflammatory material in the ethmoid, maxillary and sphenoid sinuses. She presented haemodynamic instability and vasoactive support was necessary. The diagnosis of the patient was pneumococcal meningitis and pneumococcal pneumonia. The blood culture collected in the emergency department on admission was positive for coagulase-negative pathogens, the culture of cerebrospinal fluid and the BAS were positive for *Streptococcus pneumoniae*, the serotype was 12F. Therefore, levofloxacin 500 mg/12 h was added. On the 7th day, chest X-ray revealed large con-

Table 2. Baseline characteristics, risk factors, symptoms, radiographic location, diagnostic methods and treatments of the four patients

	Patient 1	Patient 2	Patient 3	Patient 4
Sex	Male	Male	Male	Female
Age	45 y	27 y	35 y	49 y
Alcohol abuse	–	–	++	–
Smoking	–	–	++	–
Underlying disease	Bronchial asthma	–	–	Psychiatric disease
Fever	+	+	++	++
Cough	++	+	–	–
Sputum	++	+	–	–
Haemoptysis	+	–	–	–
Chest pain	–	–	++	–
Anorexia	+	+	–	–
General malaise	+	+	–	+
Radiographic location	Right upper lobe	Right upper lobe	Left lower lobe	Right lung
Pleural effusion	–	–	+	–
White blood cells	8540	10.700	14.000	19.700
Neutrophils%	74.5	75.5	84.0	76.0
Lymphocytes%	13.0	14.1	6.0	3.3
Reactive c-protein mg/dl	3.51	8.05	22.1	27.2
Blood culture	–	–	+	+
Tracheo-bronchial culture	+	+	+	+
Broncho-alveolar culture	–	–	–	–
Urinary antigen	–	–	+	–
Cerebro-spinal fluid	–	–	–	+
Antibiotic therapy	Amoxicillin/clavulanate	Amoxicillin/clavulanate	Meropenem + vancomycin +levofloxacin	Meropenem + vancomycin + levofloxacin

solidation with a cavitary lesion inside (68 mm diameter) with thickened wall and irregular inner edges (compatible with lung abscess) (Fig. 4B). BAS was performed for secretions. CT-guided puncture of the cavitation was performed, which yielded pus. The culture of the sample obtained was negative. Genetic probe of *Mycobacterium tuberculosis* was negative. The patient was extubated on the 16th day. Chest CT on the 18th day showed a large consolidation adhering to a large pleural fissure involving the right upper lobe and presenting a smaller size cavitation inside (Fig. 4C). She was transferred to the ward on the 21st day and discharged seven days later with a further reduction of fluid-filled lung cavity presenting with thickened wall and irregular edges (Fig. 4D).

The clinical data of the four patients are summarised in Table 2. Written, informed consent was obtained from the four patients for the publication of this case series and any accompanying images.

Discussion and conclusions

Diseases caused by *Streptococcus pneumoniae* are a major public health problem worldwide. Invasive diseases caused by pneumococcus include pneumonia, meningitis, bacteraemia, and pneumonia with bacteraemia, lung abscess and/or empyema [6]. Currently 46 serogroups and 93 serotypes have been documented; serotype 6C, serotype 6D, and serotype 11 are the latest additions [6]. The most severe pneumococcal disease most frequently affects individuals at extremes of age and those with immunological impairment (children or adults).

Nevertheless, a significant proportion of adults who develop invasive infection have no apparent pre-existing risk factor [7–9]. Most cases of severe pneumococcal disease are caused by a limited number of serotypes that vary in infectivity and virulence. Host and bacterial

factors both contribute to the pathogenicity. The coincidence of two key situations may lead to the development of disease (first, colonisation of the host with a pneumococcal serotype that it has not yet established immunity to, and second, an alteration of the natural barriers or host immune system) [8]. *Streptococcus pneumoniae* frequently colonises the nasopharyngeal region; spread from the nasopharynx to the lower respiratory tract or other sites may cause invasive disease. The nasopharyngeal carriage rate is highest in children, mainly during the first years of life (nasopharyngeal carriage rates range from 20% to 50% of healthy children) rather than in the adult population (nasopharyngeal carriage rates range from 5% to 30% of healthy adults) [6]. Risk factors for nasopharyngeal carriage in children include winter season, age < 6 years, having young siblings and attendance in daycare centres. In adults, risk factors for nasopharyngeal carriage include cigarette smoking, asthma, and acute upper respiratory infection [6]. In children, colonisation may persist for a mean of 4 months, but this is much shorter in adults, usually 2 to 4 weeks [10].

Transmission of *pneumococcus* from children to household contacts or adults is the principal cause of nasopharyngeal carriage and severe pneumococcal disease [6].

Not all pneumococcal serotypes are equally virulent; capsular polysaccharide is the major virulence factor, and those serotypes that produce large amounts of polysaccharide are likely to be more virulent than others. After invasion, the capsular polysaccharide also protects the pathogen by inhibiting neutrophil phagocytosis and classic complement-mediated bacterial killing. Some of the important factors in the development of pneumococcal disease include the invasive properties of the serotype, the ability of the organism to evade the immune system and the absence of a type-specific pneumococcal antibody [11]. Ethnicity (African Americans, American Indians, Native Alaskans and Australian Aborigines), extremes of age (< 2 or > 65 years), existence of comorbidities (pulmonary, neurological, hepatic, diabetes mellitus, renal conditions — especially renal insufficiency or nephrotic syndrome, alcoholism and immunosuppression — asplenia, human immunodeficiency virus, and sickle cell anaemia — anatomic abnormalities — cerebrospinal fluid leak, cochlear implant or congenital heart disease) are well-known risk factors associated with an increased susceptibility to severe pneumococcal infection and are further associated with higher mortality [6]. Asplenia and spleen disfunction are linked to specific defects in host responses due to low levels of antibodies and complement factors; the combination of these defects contributes to increased rates of more severe pneumococcal disease [7–9].

In contrast, exposure to cigarette smoke and multiple children in the household are risk factors in healthy children. In the case of immunocompetent adults, the incidence of severe pneumococcal disease is increased with the following comorbidities: congestive heart failure, chronic lung disease, asthma, diabetes mellitus and neurological disorders. Alcohol abuse, cigarette smoking, recent influenza infection, institutionalisation, male sex and black race are also risk factors in this population [6].

In the past, *Staphylococcus aureus* was frequently the aetiological agent of lung abscess. In recent years, *Streptococcus pneumoniae* has been implicated more often [12, 13]. A lung abscess is an area of suppuration and necrosis involving one or more areas of the lung parenchyma [14]. Abscesses form as a consequence of infection and destruction of the lung parenchyma with central necrosis, leading to cavity formation. The characteristic radiograph finding is an air-fluid level on the chest radiograph [14, 15]. A lung abscess may decompress and eventually resolve, if it communicates with the tracheobronchial tree; however, if no communication develops surrounding the necrotic lung, purulent material becomes thick and fibrotic. Lung abscesses can result from widely variable pathogenic processes, including necrotizing pneumonia, aspiration pneumonia, focal infection of the lung during high-grade bacteraemia, or as a consequence of septic emboli or subacute airway infection. Conditions contributing to this pathogenic process include cystic fibrosis, tracheoesophageal fistula, gastroesophageal reflux, immunodeficiency, alcohol or drug use, acute and chronic aspiration and poor dentition [14].

Pneumonia, high-grade bacteraemia or septic emboli also predispose patients to abscess formation. Necrotizing community-acquired pneumonia is the most common underlying condition (particularly in children) with lung abscess [5, 14, 16].

Lung abscess could be also classified in two groups: 1) primary lung abscesses, which occur without other underlying disease processes, and which are a direct result of pneumonia or aspiration; 2) secondary lung abscesses, which develop in those with an underlying medical

condition such as immunocompromised states, other lung diseases, central nervous system disorders and congenital heart diseases. The abscesses are located predominantly in posterior segments of the upper lobes or in superior segments of the lower lobes. They are typically singular and appear with equal frequency on the right or left side. Multiple or bilateral abscesses are rarely caused by aspiration, when a present pathogenic mechanism other than aspiration (for example, an extrapulmonary focus) should be suspected [17]. The presence of pericavitary lesions and pulmonary consolidation foci located far from a cavitary lesion suggest tuberculosis. Both clinical examination and postero-anterior coupled with lateral chest X-ray most frequently clearly identify the characteristics and location of these lesions. It is possible to visualise the air-fluid levels within the cavities [17]. Chest CT and endoscopic procedures are important in the analysis of the condition of the drainage bronchus and the abscess cavity wall, especially for making differential diagnosis of lung carcinoma [17]. A high number of patients undergo bronchoscopy, despite current recommendations, which suggest that this invasive technique should only be used in atypical cases and/or in those with a high degree of suspicion of mechanical obstruction [18, 19]. The duration of antibiotic administration in previous reports was estimated to be from 28 to 48 days and it was consistent with our results [1, 18, 20, 21].

A recent study in an adult population, concerning pulmonary complications of pneumococcal pneumonia, showed that 38% of this cohort had pulmonary complications (e.g. necrotizing pneumonia and empyema). Patients with pulmonary complications were younger, had less comorbidity and a longer length of hospital stay, more frequent admission to ICU, but a mortality rate similar to uncomplicated pneumonia [22]. Our four patients (although they presented different clinical pictures) were all young, and two of them had no comorbidity; all of them had a long hospital stay and none died. The first two patients had an onset of clinical symptoms with low grade fever, asthenia, anorexia and weight loss and absence of typical pneumococcal infection symptoms. In these cases diagnostic procedures must be considered for a differential diagnosis of lung cancer, pulmonary tuberculosis, pulmonary sarcoidosis, pulmonary haematoma and Wegener's pulmonary vasculitis [23]. While the majority of pneumococcal pneumonia cases are non-bacteraemic (60–80%), bacteraemic pneumonia is generally more severe [22]. The patients in the last two cases showed the presence of bacteria in blood cultures, and one of them also in cerebrospinal fluid. They were admitted to intensive care. Also, the serotypes found in our cases were extremely rare compared to most cases of pneumonia and/or pulmonary abscess [4, 24, 25]. The same is true in the patient with spinal meningitis [24, 25]. To our knowledge, this is the first time that 12F *Streptococcus p.* has been reported as the cause of pulmonary abscess. The incidence of pneumococcal pneumonia is greatest at the extremes of age and in individuals with medical comorbidity, but its complications usually involve young and healthy patients. Pneumococci colonise the nasopharynx and from there can spread directly via the airway to the lower respiratory tract, causing lower infectious disease. The ongoing evolution of pneumococci will continue to challenge researchers and clinicians; areas of future attention will be discovering early stages of the disease. Such knowledge could be useful to better stratify patients at risk [26].

Conflict of interest

The authors declare no conflict of interest.

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